Medical History

Infantile scurvy: the centenary of Barlow's disease

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Scurvy. A disease characterised by general debility of the body, extreme tenderness of the gums, foul breath, subcutaneous eruptions and pain in the limbs, induced by exposure and by a too liberal diet of salty foods. (*Oxford English Dictionary*, 1979)

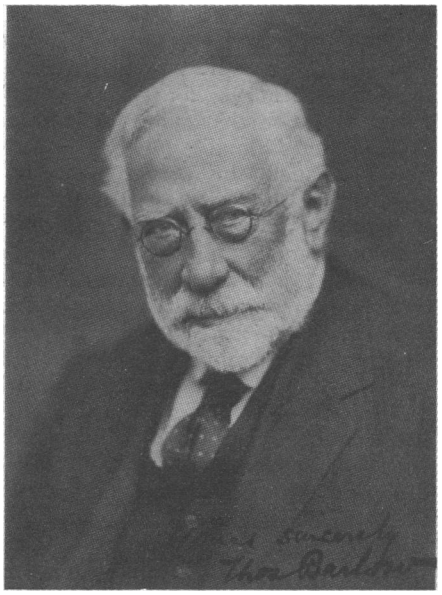
"Scurvy" is a folk word that first appeared in the Middle Ages. Later long sea voyages led to the connection of the disease with prolonged exposure and salty foods, as the reference above suggests. Jacques Cartier's sailors, exploring lower Canada in 1536, are known to have suffered severely from scurvy.[1] In an attempt to find the cause of the illness the corpse of one of his sailors who died from the disease was dissected, but this did not shed any light on the cause of the disease.[2] But a cure was suggested to Cartier by a native chief who told him to make a decoction of the bark and leaves of a tree (possibly the American spruce). This cured the sailors within six days but was probably not the first cure known to the West, for "scurvy grass" (*Cochlea officianalis*) is an old name.[3]

The name of James Lind is closely linked with scurvy. Lind was born in Edinburgh in 1716 and served as a surgeon's mate in the Royal Navy. He saw many cases of scurvy during his nine years at sea, and after leaving the service and graduating as an MD in Edinburgh he published his "treatise of the scurvy."[1] This proposed that in people predisposed to scurvy "an additional, and extremely powerful cause observed at sea was . . . the want of fresh vegetables and greens." He showed that the juice of oranges or lemons was both curative and preventive, and strongly recommended that this should be given routinely to all sailors. Despite his intensive campaign the Admiralty did not take up his suggestions until a year after his death in 1794, when lemon juice was added to sailors' rations.

Another name in the history of scurvy is John Huxham of Plymouth, who wrote a letter in 1747 emphasising the importance of "fruits and garden-stuff" in preventing scurvy, but his letter was not published until 10 years later[4] and his book *de Scorbuto* appeared later still, in 1766. Huxham came from Devon and not unnaturally favoured apples as an antiscorbutic remedy. He suggested that "every sailor should have at least a pint of Cyder a day, besides Beer and Water."

By the beginning of the nineteenth century scurvy should have been a curable and preventable condition. But the authorities were amazingly slow to implement the suggestions of Lind and Huxham. On Scott's expedition in 1901 Shackleton developed scurvy and was said to be "not much impressed with the toxic theory but showed a greater interest in the value of fresh food."[5] It is interesting to observe that even the 1979 definition quoted at the head of this article could have been written 200 years ago.

In the face of such persistent ignorance it is not surprising that physicians failed to recognise scurvy in children. Still said that Glisson described scurvy in children in 1650, but no further mention was made until some 200 years later.[6] This is unsurprising, as Glisson used the words "scurvy" and "scorbutus" as alternative terms for "lues venerea."[7 8] As late as 1889 there was no mention of scurvy in Henoch's textbook of paediatrics, although in discussing rickets he stated that he had encountered a few patients with a scorbutic condition of the gums.[9]



Portrait of Sir Thomas Barlow.

Scurvy in children presented a different picture from that in adults. The infant was alert but motionless from fear of pain and screamed when his cot was approached or he was handled. There was swelling about the knees and thickening of some bones, and purple swelling of gums only after teeth had erupted, but there were no bruises, dyspnoea, or phrynodermia.

During the second half of the nineteenth century the condition of acute rickets was described. In 1859 and 1862 Moeller of Königsberg (now Kaliningrad)—a city where learning was so revered that the inhabitants rang the bells when Kant recovered from a cold[10]—described two cases of "acute rickets which reminded one decidedly of an osteitis." One child had swelling of the gums "very nearly like a complete scorbutic state," and Moeller excluded scurvy because antiscorbutic treatment was ineffective, but after months of illness the child recovered in the warm weather and fresh air of early summer, when incidentally given lemon juice and fresh vegetables. His second patient had "bone-bleeding" with a bloody nasal discharge and proptosis, more suggestive of neuroblastoma than scurvy.[11] In Germany the

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condition was later called Moeller-Barlow disease, but Moeller never made the mental jump required to say "This is really scurvy." In 1871 Ingerslev, a young medical assistant in Copenhagen, got it right.[12] He described a 15 month old child and diagnosed scurvy without mentioning acute rickets. He went on to say that the cause was lack of vegetables and described the use of lemon juice and raw vegetables in treatment. The child had been fed on Liebig's Baked Infant Food, and increased use of such manufactured foods was, presumably, why infantile scurvy became such a problem.

Ingerslev's report was in Danish and thus was not read by Mr Tom Smith, Dr Cheadle, or Dr Barlow, three key figures in the recognition of childhood scurvy in England. Smith was a surgeon at St Bartholomew's Hospital and at the Hospital for Sick Children, Great Ormond Street, London. His name persists in "Tom Smith's disease"—disappearance of the head of the femur due to infection in childhood. Barlow and Cheadle were both physicians who also practised at Great Ormond Street. In 1876 Smith, using notes written by Barlow, described a fatal case of haemorrhagic osteitis.[13] Barlow initially agreed with his diagnosis but changed his mind later.[14]

In 1881 Cheadle described three children who, in his opinion, suffered from rickets and scurvy. They had not been exposed to arctic cold or tropical heat or been engaged in severe exertion, but they had been "strangely dieted . . . the diet was more than a rickety diet, it was a *scurvy* diet." He believed that the children of the poor did not usually have scurvy because their parents could not afford farinaceous feeds but used potato instead. In treatment he used potato, fresh milk, and orange juice. Clearly he was on the right tack, but scurvy was not called Cheadle's disease because he left the impression that he was describing scurvy and rickets, not just scurvy, and he did not cover the subject as comprehensively as Barlow.

At a meeting of the Medico-Chirurgical Society of London on 27 March 1883 Thomas Barlow read a paper on "Cases described as 'acute rickets' (combination of rickets and scurvy)." In his title he repeated Cheadle's error in suggesting that the disease was a combination of scurvy and rickets, but the expanded title of his complete text in the transactions of the society makes his position clear: "On cases described as 'acute rickets' which are possibly a combination of rickets and scurvy, the scurvy being essential and the rickets variable."[14]

Barlow first "narrated a typical case" in a child $1\frac{1}{2}$ years old, and went on to analyse the symptoms in 12 other cases, adding 19 reported by other authors. The symptoms that he originally described, and later repeated in his Bradshaw Lecture in 1894, were: a sudden onset in a child over 4 months old, pallor, adequate subcutaneous fat, screaming when the legs were handled, pseudoparalysis, swelling of limbs, crepitus due to fracture in or near epiphyses, occasionally proptosis, spongy gums (where teeth had erupted) with foetor and bleeding, deformities of ribs, osseous sheaths surrounding bones, and albumin and blood in urine; nothing of note has since been added to his clinical observations.[15] Knowledge of the disease was later disseminated by an excellent account in the first edition of Osler's textbook of medicine.[16] In 1897 the Surgeon General's Catalogue listed 54 references to Barlow's disease.

Barlow demonstrated the pathological changes of scurvy by bringing specimens of bones to the meeting, and these may still be seen in the Great Ormond Street museum, now housed in the Institute of Child Health, Guilford Street, London. They are unusually vivid for old specimens, carmine having been used to simulate fresh blood. Barlow also examined a histological preparation which supported his opinion that the condition was not caused by inflammation.

His achievement lay in his clear exposition of the disease and its cause and cure. He noted that breast fed babies did not get scurvy; meat and fresh cows' milk were antiscorbutic but less so than vegetables; the antiscorbutic power was destroyed by heating. Like Cheadle he said that scurvy occurred in the children of well to do people living in good homes in good surroundings.

Barlow was only 37 when he made these statements about scurvy, and he went on to become a famous clinician. He was president of the Royal College of Physicians and physician extraordinary to Queen Victoria, King Edward VII, and King George V. He was renowned for his vast knowledge, kindliness, and genuine sympathy. His hobbies were botany, geology, art, and music. On his 90th birthday he was presented with a special number of the *Archives of Disease in Childhood* devoted exclusively to the condition that he had described more than half a century before.

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(*Accepted 8 September 1983*)

A girl aged 10 is profoundly deaf as a result of intrauterine rubella. Her mother questioned whether she needed to have rubella vaccination. A blood test showed that she is not, however, immune. Presumably her immune system was not capable of forming antibodies when she was exposed to the infection. Should I give my patient rubella vaccine and check that she becomes immune subsequently?

Certainly it would be advisable to immunise this patient with rubella vaccine if she is susceptible. It is policy to vaccinate schoolgirls between the ages of 11 and 13 without first testing for antibodies, although at this age 60-65% will already be immune through previous infection. As it is usual to find specific antibodies in congenital rubella three possibilities should be considered. Firstly, a misidentification at some point in the chain from taking the sample to issuing the report could have occurred. Analysis of a repeat specimen would settle this. Secondly, rubella antibody screening is done at a set level of activity below which samples are reported as not immune. These will include patients with very low levels of antibody as well as frank negatives. The level most laboratories choose errs on the side of reporting low levels of antibody as non-immune on the premise that it is better to vaccinate a small percentage of patients unnecessarily than to miss a susceptible patient. Inquiry at the laboratory may show that the patient has trace amounts of rubella antibody. The third point to consider is the history. Was there clinical evidence and serological confirmation of rubella infection during the mother's pregnancy? Are there other manifestations of congenital rubella than deafness in the patient, and were virological tests done to establish this after her birth? If, however, this information is lacking and her serum contains no trace of rubella antibodies the practical solution is to immunise her and test for seroconversion by taking another specimen three months later. Clearly, it is important to allay the mother's anxiety by proving that her child is protected.—A A CODD, consultant virologist, Newcastle upon Tyne.